Pacemaker-Induced Coherence in Cortical Networks

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A simple mathematical model of cortical tissue is introduced and the system's dynamics is monitored when a small subset of neurons is submitted to oscillatory inputs of various frequency and wave form. In the absence of input, the system shows desynchronized or "turbulent" behavior. The oscillatory input synchronizes the neuronal activity, which is strongest for inputs of low frequency. The increase of spatial coherence is estimated from the spatial autocorrelation function whereas the increase in temporal coherence is evaluated from correlation dimensions. The model accounts qualitatively for some of the features of the thalamocortical system.

1 Introduction

The electroencephalogram (EEG) of an aroused and active brain is characterized by irregular beta waves of high frequency and low amplitude. As the eyes are closed and the subject is relaxing, more synchronized alpha waves appear. As the subject drifts toward sleep, the brain enters into a succession of four stages where the EEG coherence and amplitude gradually increase as the mean frequency decreases. In a normal brain, the fourth sleep stage — or deep sleep — is characterized by the highest EEG amplitude, while the mean frequency of oscillations is the lowest.

The discovery of massive interconnections between thalamus and neocortex as well as the ability of the thalamus to produce periodic oscillations even when disconnected from the cortex led to the "thalamic pacemaker" hypothesis for the generation of brain rhythms (Andersen and Andersson 1968; Steriade and Deschênes 1984). Thus it seems that the behavioral states of the brain are somehow related to an oscillatory input into the cortical tissue.

The object of this paper is to introduce a simple mathematical model to investigate the influence of an oscillatory input on a cortical tissue. A very small percentage of cortical cells is submitted to inputs of increasing frequency as well as different wave forms. The degree of synchrony of the network as a result of the input is investigated by evaluating the spatial autocorrelation function.
Reminiscient of EEG activity which is an average property of neural masses, the spatial average of the network activity is monitored in time. The temporal coherence of this mean activity is assessed with the help of techniques from nonlinear time series analysis. The mean activity shows a deterministic chaotic dynamics, which may be quantified by evaluating correlation dimensions. The numerical value of the correlation dimension characterizes the coherence of the system.

The properties of the model are compared with the various dynamical parameters evaluated from the human EEG.

2 The Model

In the cortical tissue, the population of neurones is divided into approximately 80% excitatory and 20% inhibitory neurones (Braitenberg 1986). The dynamics of such a tissue is described in terms of the electrical analogue of the neuronal membrane (Hodgkin and Huxley 1952) and reads:

\[
\frac{dX_i}{dt} = -\left(\Delta X_i - Y_i - F_i\right) - \Delta Y_i - \omega_{ji}^{(1)} \left(\sum_k \omega_{ki}^{(1)} F_k[t - \tau_{ki}]\right) \\
+ T_i g(x) \\
- (X_i - E_2) \sum_l \omega_{li}^{(2)} F[Y_l(t - \tau_{li})]
\]

\[
\frac{dY_j}{dt} = -\gamma(Y_j - V_0) - (Y_j - E_1) \sum_k \omega_{kj}^{(3)} F[X_k(t - \tau_{kj})] \\
- (Y_j - E_2) \sum_l \omega_{lj}^{(4)} F[Y_l(t - \tau_{lj})]
\]

\[i, k = 1, \ldots, N, \quad j, l = 1, \ldots, M\]  \hspace{1cm} (2.1)

\(X_i\) and \(Y_j\) are the postsynaptic potentials of the \(N\) excitatory and \(M\) inhibitory neurones, \(\gamma = 0.25\) msec\(^{-1}\) is the inverse of the time constant of the membrane and \(V_0 = -60\) mV is the resting potential. \(E_1 = 50\) mV and \(E_2 = -80\) mV are the excitatory and inhibitory transmitter equilibrium potentials. \(\omega_{ji}^{(1)}, \omega_{ji}^{(2)}, \omega_{ji}^{(3)} \omega_{ji}^{(4)}\) are, respectively, the excitatory-to-excitatory, inhibitory-to-excitatory, excitatory-to-inhibitory and inhibitory-to-inhibitory synaptic weights. \(F(x) = 1/(1 + \exp[-0.2(\sigma + 25)])\) describes the nonlinear properties of neurones such as the firing threshold and the saturation of the firing rate. \(\tau_{ji}\) is the propagation delay between sites \(i\) and \(j\) of the network.

The two types of neurones are arranged on a two-dimensional regular lattice and \(\tau_{ji}\) is 2 msec per lattice length. A given neurone connects to all
For each type of interaction, the synaptic weights of all neurons are identical and the total sum of the inputs is constant. Therefore, $\sum_k w^{(1)}_{ki} = \Omega_1$, $\sum_i w^{(2)}_{ii} = \Omega_2$, $\sum_k w^{(3)}_{kj} = \Omega_3$, and $\sum_i w^{(4)}_{ij} = \Omega_4$. The dynamic properties of the model are very similar for a wide range of these parameters.

A small fraction of cortical excitatory neurons is submitted to an oscillatory input that mimics thalamic activity. A phenomenological model of the thalamic oscillations was introduced by Rose and Hindmarsh (1984).

The two variable version of the model reads:

$$\frac{1}{c} \frac{dx}{dt} = f_1(x) - z + I$$

$$\frac{1}{c} \frac{dz}{dt} = r[h_2(x) - z] \tag{2.2}$$

where $x$ is the membrane potential and $z$ is an adaptation current. The details of the model can be found in Rose and Hindmarsh (1984) (see Fig. 5 of this paper for the parameter values).

This model accounts qualitatively for some of the electrophysiological properties of thalamic neurons such as the coexistence between resting and oscillating activities. $I$ is the control parameter allowing the transition from steady states to oscillatory states. $c$ is a parameter which rescales the time axis.

The thalamic oscillator is connected to 2% of randomly chosen excitatory neurons via the function $g(x) = 1/[1 + \exp[-10(x - 0.9)]]$ with a strength of $T_i=15$. This input is identical for all receiving cortical neurons.

The set of equations 2.1 together with equations (2.2) are integrated numerically using a Runge–Kutta algorithm modified for integration of delay differential equations.

3 Spontaneous Dynamics of the Network

We first investigate the behavior of the cortical network in the absence of input ($T_i=0$). The activity of network is a function of the total synaptic weights $\{\Omega_1, \Omega_2, \Omega_3, \Omega_4\}$. For weak values of these parameters, all cells of the network relax to the unique resting potential of $-60$ mV. However, as the synaptic weights increase, spontaneous sustained activity may appear.

For $\Omega_2 = \Omega_3 = 12.5$ and $\Omega_4 = 0$ and for moderate values of $\Omega_1$, the neurons oscillate periodically in unison and one sees a bulk oscillation. As $\Omega_1$ is increased further, spatiotemporal chaos may appear. In the range of parameters considered, this type of “turbulence” is not seen in small networks. However, for a reasonably large number of neurons ($N \sim 400$), turbulence is always present.
Between the oscillating regime and the turbulent state, there is a range of values of $\Omega_1$ where intermittency is seen. Periods of oscillatory regime alternate with spatiotemporal chaotic activity. For still larger values of $\Omega_1$, spiral waves may be seen.

The activity of neurons during turbulence is seen as an irregular propagation of clusters of activity (Fig. 1a). Such clusters spontaneously appear and collide to form very complex patterns. Sometimes transient spiral waves may also appear. Individually, each neuron behaves as an aperiodic oscillator.

4 Spatial Aspects of Synchronization

The onset of oscillatory input generally changes the global activity of the network. Although a small minority of cells receive the input, the vast majority of the neurons may be entrained into a coherent behavior.

We shall investigate the dynamics of the system for two values of the input $I$ and parameter $c$, $\{I = 0.6, c = 0.06\}$ and $\{I = 2.6, c = 0.125\}$, which correspond to two oscillating states of high and low frequency of different amplitude and wave form. The pacemaker oscillations used are illustrated in Figure 1b–1c.

In Figure 1b, the system is submitted to the faster rhythm of the pacemaker. From a desynchronized state with $T_i = 0$, the network switches into a partially synchronized dynamics where the activities of the neurons become more phase-locked. As the pacemaker oscillates, the network also oscillates between various spatially synchronized patterns. Figure 1b is an instantaneous snapshot that changes at every moment. However synchronized states very similar to Figure 1b appear very often. Although these oscillations are not strictly regular, the mean frequency of the network is of the same order as that of the pacemaker.

If the parameter $I$ is decreased, the pacemaker switches to a slower oscillating regime. Again the network responds by a spatial synchronization of neural activity. Similar snapshots to Figure 1b are also seen in this case. In addition, the system shows brief episodes of a remarkable degree of coherence, as illustrated in Figure 1c. These brief occurrences of almost total synchronization appear irregularly.

The level of synchronization between neurons can be quantified by evaluating the spatial autocorrelation function. Figure 2 represents this function evaluated for the system in the absence of input and for slow and fast pacemaker inputs. For the turbulent state, this function vanishes after several lattice lengths, indicating a loss of spatial coherence and the absence of long-range spatial correlations. However, as the fast oscillatory input is switched on, the dynamics of the network synchronizes and the autocorrelation function shows a slower spatial decay. During this state, long range spatial correlations appear as a consequence of the more coherent synchronized dynamics. Spatial correlations increase further for the slow periodic input.
Figure 2: Spatial autocorrelation function of the network. Solid, "turbulent" state in the absence of pacemaker; dashed, fast pacemaker input; dotted, slow pacemaker input. The parameters are the same as in Figure 1.

There is an obvious oscillation of a period of two lattice lengths superimposed on the correlation function (Fig. 2). This fact indicates that the neighboring excitatory cells have a tendency to oscillate out of phase.

5 Temporal Aspects of Synchronization

The degree of relative temporal coherence of the network may also be studied by examining the evolution of the spatially averaged membrane potentials in function of time. Such an average activity constitutes a global variable which may be thought as being the analogue of an EEG. Figure 3 shows the time evolution of these average values in the absence and in the presence of fast and slow oscillatory inputs. To save computer time, a smaller network was considered as qualitatively it shows similar behavior to the larger network. In the absence of pacemaker, we observe fast and irregular behavior. However, as the periodic input sets in, large amplitude and more regular slower oscillations are seen. The slow pacemaker oscillation organizes the system into a higher amplitude and lower frequency region.
Figure 3: Spatially averaged membrane potentials. (a) Desynchronized "turbulent" synchronization during the slow rhythm. Vertical calibration bars are of 50 mV. TI \( \tau = 6 \) delay \( \tau \) used for phase space reconstruction is of 6 msec. The parameters are the same as in Figure 1, except \( N=400 \) and \( M=10 \).
Figure 3 shows the phase portraits constructed from the three time series. It is seen that in the absence of pacemaker, no obvious attractor exists. However, the oscillatory input organizes the system such as the averaged behavior shows deterministic chaos. The numerical values of the correlation dimension of these attractors are convenient parameters for assessing the degree of synchronization of systems described in Figure 3.

In the absence of pacemaker activity, we are in the presence of a very high dimensional dynamical system, and the existing algorithms do not permit the characterization of the system.

As the pacemaker activity sets in, the collective oscillating behavior of the network is characterized by low dimensional chaos. The correlation dimension is of $6.6 \pm 0.1$ for the faster rhythm and of $3.6 \pm 0.1$ for the slower rhythm.

The correlation dimension has also been calculated for various values of the time scaling parameter $c$, with parameter $I$ constant. The corresponding pacemaker oscillations are therefore identical in wave form, however show a decrease in frequency. Figure 4 shows that in this case, the correlation dimension increases *linearly* with the pacemaker

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**Figure 4:** Correlation dimension vs. pacemaker frequency. $N = 400$, 10,000 data points equidistant of 1.2 msec are used (about 80 to 320 pseudoperiods). The correlation dimension is estimated using the Grassberger–Procaccia algorithm and the time delay $\tau$ is estimated from a check for stationarity: 6 msec $\leq \tau \leq$ 12 msec. (●) Same oscillator ($I = 0.6$) rescaled with $c = 0.06$, $c = 0.125$, $c = 0.185$, and $c = 0.25$. (○) 26 Hz oscillation of different wave form ($I = 2.6$, $c = 0.125$).
frequency. For very slow thalamic inputs, the time series looks progressively more coherent and have a correlation dimension between 2 and 3.

6 Discussion

Experimental data point to the important role of thalamic oscillations in relation to the various behavioral states. On the other hand, the nonlinear time series analysis of EEG, which result from the averaged behavior of neural masses, has shown a decrease of correlation dimension (Babloyantz and Destexhe 1987; Babloyantz et al. 1985; Destexhe et al., 1988).
References


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